COMPARATIVE RESPONSE OF SWINE AND RATS TO HIGH-FIBER OR HIGH-PROTEIN DIETS[1]

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ABSTRACT

Twenty-four growing swine and 24 growing rats were fed high-protein (34%) diets on an ad libitum basis to determine their effects on body weight, carcass characteristics, intestinal microbiological profile and visceral organ weights. High dietary fiber reduced body weight gain and gain:feed ratio in both swine and rats and decreased body fatness in swine; it increased relative kidney weight (percentage of body weight) in both swine and rats and decreased relative liver weight in rats but increased it in swine. Absolute weights of stomach and large intestine were unaffected by high fiber in either species, but relative weight of small and large intestine was increased in swine and relative weight of stomach was increased in rats. High dietary protein increased absolute and relative weights of kidneys in both species and increased relative liver in swine but not in rats. Absolute weight of large intestine was increased by high dietary protein in rats and tended to be increased in swine; relative large intestine weight was increased in both species. The microbial profile of large intestinal contents of rats showed no effect of diet on Enterobacteriaceae, Campylobacter, Salmonella or total anaerobes and cellulolytic organisms, but coliforms were higher in rats fed high fiber or high protein than in controls. We conclude that dietary levels of fiber and protein influence growth of specific segments of the gastrointestinal tract of growing rats and swine, probably by different mechanisms of action.
(Key Words: Dietary Fiber, Proteins, Gastrointestinal Tract, Microflora, Pigs, Rats.)

Introduction

Response of the gastrointestinal tract to diet intake and composition has been addressed recently (Koong et al., 1982a,b, 1983, 1985; Pekas et al., 1983; Ferrell and Koong, 1986;

Pond et al., 1986). Visceral organ mass, which makes up 10% or less of total body mass, accounts for a disproportionate share of body heat production. For example, Webster (1981) estimated that heat production of the liver and gut represented about 20% of the fasting heat production of rats at rest. Fasting heat production was highly correlated ($r = .90$) with visceral organ weights (Koong et al., 1982a,b, 1985).

This relationship between visceral organ size and basal heat production has important implications for food animal production, because nutrients are diverted from the growth of the edible carcass when visceral organ weights are increased. The purpose of the present experiments with rats and swine was to ascertain the relative effects of high-fiber or high-protein diets on body weight gain, feed utilization, organ weights and large intestine

TABLE 1. COMPOSITION OF DIETS

Ingredient	Control	High fiber	High protein
Corn (No. 2 yellow)	82.1	51.9	26.1
Alfalfa meal		40.0	
Soybean meal (44% CP)	14.0	6.0	70.0
Dicalcium phosphate	2.4		2.4
Monosodium phosphate		1.1	
Limestone	.5		.5
Salt (iodized)	.4	.4	.4
Trace mineral premix[a]	.2	.2	.2
Vitamin premix[b]	.2	.2	.2
Choline chloride	.2	.2	.2
Total	100.0	100.0	100.0
Analyzed composition			
DM, %	89.7	89.6	90.3
CP, %	14.6	16.4	34.1
ADF, %	4.5	16.2	7.6
Cell walls, %	10.0	24.9	10.1
Cellulose, %	3.7	12.5	6.7
Lignin, %	1.3	4.3	1.5
Ash, %	5.7	6.8	7.1

[a]Supplied the following (units/kg of complete diet): Cu, 10 mg (as $CuSO_4 \cdot 5H_2O$); Fe, 160 mg (as $FeSO_4 \cdot 7H_2O$); Mn, 20 mg (as MnO); Zn, 100 mg (as ZnO); $CaCO_3$ as a carrier.

[b]Supplied the following (units/kg of complete diet): vitamin A, 5,280 IU; vitamin D_3, 704 IU; dl-alpha-tocopheryl acetate, 32.5 mg; menadione bisulfate, 3.52 mg; vitamin B_{12}, 26.4 mcg; riboflavin, 5.28 mg; niacin, 28.16 mg; d-pantothenic acid, 21.12 mg; biotin, 88 mcg; thiamin, 2.2 mg.

microbial populations (rats only) in view of evidence indicating that high fiber (Bohman et al., 1955; Kass et al., 1980; Pekas et al., 1983) and high protein (Pond et al., 1986) are associated with gastrointestinal trace hypertrophy and that cellulolytic bacteria are increased in the large intestine of swine fed high fiber (Varel, 1987).

Materials and Methods

Swine Experiment. Twenty-four crossbred (Chester White × Landrace × Large White × Yorkshire) castrated male finishing pigs (avg body weight, 93 kg) were divided into three groups of eight and assigned randomly to three diets as follows (see Table 1 for composition): control (C,corn-soybean meal fortified with minerals and vitamins); high fiber (HF,40% alfalfa meal substituted for part of corn and soybean meal) and high protein (HP,soybean meal replacing 56% of the corn diet to provide a 34% protein diet).

Pigs were allowed to consume their respective diets ad libitum and were kept in pairs (four pens of two pigs per diet) in 1.8-m^2 concrete, slotted-floor pens in a temperature- and light-controlled building (20 ± 2°C, 12 h light:12 h dark cycle). Body weight and feed consumption of each pen of pigs were recorded on d 0, 28, 56 and 84. All pigs were slaughtered on d 84 without fasting. Carcass weight; length; backfat depth (mean of three measurements taken on the split carcass at the first and last ribs and last lumbar vertebrae); cross-sectional area of longissimus muscle and of s.c. fat at the 10th-11th rib interface; weight of untrimmed and trimmed Boston butt, picnic, loin and ham on the left side of the carcass; liver and kidney weight; and weight of empty stomach, small intestine and cecum-colon were recorded. Sections of duodenum, jejunum, ileum and colon from six pigs in each diet group were fixed in 10% neutral buffered formalin for histopathological examination. Tissues were embedded in paraffin, sectioned at 4 to 6 microns and stained with hematoxylin and eosin; selected sections also were stained with the Warthin-Starry stain to confirm the presence of Campylobacter. All data were subjected to analysis of variance (SAS, 1985); differences between means were tested by Least Significant Difference. Pen was the experimental unit for gain and feed data and individual animal for carcass data.

Rat Experiment. Twenty-four male Sprague-Dawley rats (avg body weight 64 g) were assigned randomly (eight rats/diet) to the

TABLE 2. EFFECT OF DIETARY FIBER AND PROTEIN ON PERFORMANCE OF
FINISHING SWINE (LEAST SQUARES MEANS)

Trait	Diet[a]			SD	Probability of diet effect
	Control	High fiber	High protein		
No. of animals	8	8	8		
Daily gain, g[b]	379[c]	239[e]	297[d]	31	< .01
Daily feed, g	1,496[c]	1,288[d]	1,263[d]	88	< .01
Gain:feed ratio	.254[c]	.185[d]	.241[c]	.019	< .01
Backfat, mm[f]	22.3[c]	18.5[e]	20.3[d]	.8	< .03

[a]See Table 1 for ingredient composition. Control = 14% protein, 0 alfalfa meal; high fiber = 14% protein, 40% alfalfa meal; high protein = 34% protein, 0 alfalfa meal.

[b]Initial weights of control, high fiber and high protein groups were 92.9, 93.0 and 92.8 kg, respectively.

[c,d,e]Means in the same row without a common superscript differ ($P < .05$).

[f]Initial backfats of control, high fiber and high protein groups were 19.8, 18.4 and 19.6 mm, respectively.

same three diets used in the pig experiment (Table 1) and fed on an ad libitum basis for 14 d. Rats were kept in a temperature- and light-controlled room in individual stainless steel wire bottom cages, each equipped with a porcelain feed dish and nipple waterer. Body weight was recorded on d 0, 7 and 14 and total feed intake was recorded. On d 14 rats were killed with diethyl ether, and liver, kidney and empty stomach, small intestine and large intestine weights were recorded. Empty stomach, small intestine and large intestine from four animals in each treatment group were oven- dried to a constant weight at 65°C to determine dried organ weight and percentage of dry matter.

A sample of colon contents was taken from four animals in each treatment group for microbiological enumeration of coliforms, Enterobacteriaceae, E. coli, campylobacter, total anaerobes, and cellulolytic bacteria (Varel and Pond, 1985). Composite samples were prepared by pooling the intestinal contents of two animals. Coliform and Enterobacteriaceae counts were performed by the pour plate method (Busta et al., 1984), using Violet Red Bile agar and Violet Red Bile Glucose agar, respectively. The plates were incubated at 37°C for 24 h. E. coli was enumerated by a three-tube most-probable-number method similar to that described by Mehlman et al.(1984). MacConkey agar plus 1% lactose was substituted for Levine's eosin-methylene blue agar. Confirmation was based on positive citrate reactions, Campylobacter was enumerated by the spread plate method (Busta et al., 1984) using the Campylobacter agar kit blaser, with

the addition of 10% porcine blood. The plates were incubated at 43°C under a modified atmosphere in GasPak anaerobe jars. The isolation and identification of salmonellae was carried out according to the method described by Andrews et al. (1984).

All body weight, feed and organ weight data were subjected to analysis of variance (SAS, 1985), differences between means were tested by Least Significant Difference. Individual animal was the experimental unit.

Results

Swine Experiment. Daily gain and daily feed consumed were depressed ($P < .01$) by high levels of either fiber or protein in the diet (Table 2). The reduction in daily feed consumed, compared with controls, was similar in pigs fed HF and HP. Daily gain was reduced more ($P < .01$) by HF than by HP, resulting in gain:feed ratios that were less than C values ($P .01$) in pigs fed HF, but not in those feed HP. Pigs fed HP had less backfat ($P < .01$) than controls, and pigs fed HF had less ($P < .01$) than those fed HP.

High dietary fiber resulted in a lower carcass weight ($P< .04$), a smaller longissimus muscle cross-sectional area ($P < .03$), a high percentage of trimmed lean cuts in the body ($P < .06$) and lighter trimmed loin ($P < .03$) compared with the controls (Table 3). High dietary protein had no effect on carcass traits, except for a smaller trimmed loin weight ($P < .03$) and a smaller cross-sectional area of s.c. fat at the 10-11th rib interface ($P < .03$) compared with the controls. Differences in

TABLE 3. EFFECT OF DIETARY FIBER AND PROTEIN LEVELS ON CARCASS
MEASUREMENTS OF FINISHING SWINE (LEAST SQUARES MEANS)

Trait	Diet[a]			SD	Probability of diet effect
	Control	High fiber	High protein		
No of animals	8	8	8		
Body wt, kg	125.8	115.6	121.6	6.9	NS
Trimmed:					
Boston butt, kg	4.34	3.97	4.33	.24	NS
Boston butt, % of BW	3.46[b]	3.43[b]	3.57[c]	.16	< .06
Ham, kg	8.30	7.90	8.11	.31	NS
Ham, % of BW	6.60	6.85	6.67	.23	NS
Picnic, kg	3.77	3.50	3.72	.18	NS
Picnic, % of BW	3.00	3.09	3.06	.17	NS
Loin, kg	8.15[b]	7.30[c]	6.95[c]	.46	< .03
Loin, % of BW	6.47	6.31	5.73	.40	NS
Lean cuts, % of BW	52.8[b]	56.3[c]	54.2[b]	1.6	< .06
Longissimus muscle, cm^2	39.9[b]	36.7[c]	40.0[b]	1.8	< .08
Longissimus fat, cm^2	66.6[b]	48.2[c]	52.1[c]	7.5	< .03
Longissimus muscle and fat, cm^2	106.5[b]	84.9[c]	92.1[b]	8.5	< .03
Cold carcass wt, kg	93.9[b]	81.9[c]	86.1[b]	4.9	< .04
Length, cm	83.4	83.2	83.4	2.0	NS

[a]Control = 14% protein, 0 alfalfa meal; high fiber = 14% protein, 40% alfalfa meal; high protein = 34% protein, 0 alfalfa meal.

[b,c]Means in the same row without a common superscript differ (P < .05).

[d]NS = not significant.

trimmed lean cut weights were related to body weight and were expressed as percentages of body weight.

High fiber had no effect on absolute or relative stomach weight (Table 4). However, relative weights (percentage of body weight) of small intestine (P < .03), large intestine (P < .10), liver (P < .01) and kidneys (P < .01) were increased compared with control values. High protein also increased relative weights of small intestine (P < .03) and large intestine (P < .10) compared with controls. Effects of HP and HF

TABLE 4. EFFECT OF DIETARY FIBER AND PROTEIN LEVELS ON ORGAN WEIGHTS
OF FINISHING SWINE (LEAST SQUARES MEANS)

Trait	Diet[a]			SD	Probability of diet effect
	Control	High fiber	High protein		
No. of animals	7[b]	6	7		
Stomach, g	555	618	607	85	NS[f]
Stomach, % of BW	.445	.532	.498	.063	NS
Small intestine, g	1,095	1,307	1,228	110	NS
Small intestine, % of BW	.853[c]	1.124[d]	1.012[d]	.081	< .03
Large intestine, g	1,339	1,600	1,787	274	NS
Large intestine, % of BW	1.041[c]	1.379[d]	1.468[d]	.201	< .10
Liver, g	1,521[c]	1,642[c]	1,950[d]	112	< .01
Liver, % of BW	1.220[c]	1.417[d]	1.603[e]	.087	< .01
Kidneys, g	302[c]	301[c]	400[d]	24	< .01
Kidneys, % of BW	.242[c]	.259[d]	.329[e]	.018	< .01

[a]Control = 14% protein, 0 alfalfa meal; high fiber = 14% protein, 40% alfalfa meal; high protein = 34% protein, 0 alfalfa meal.

[b]Means for small and large intestine are for six animals in control group.

[c,d,e]Means in the same row without a common superscript differ (P < .05).

[f]NS = not significant.

TABLE 5. EFFECT OF DIETARY FIBER AND PROTEIN LEVELS ON PERFORMANCE AND ORGAN WEIGHTS OF GROWING RATS (LEAST SQUARES MEANS)

Trait	Diet			SD	Probability of diet effect
	Control	High fiber	High protein		
No. of rats	8	8	8		
Initial wt, g	62.0	64.3	65.8	3.3	NS[d]
Final wt, g	135.1[a]	106.8[b]	140.7[a]	10.0	< .01
Daily gain, g	5.2[a]	3.0[b]	5.4[a]	.6	< .01
Daily feed, g	17.9[a]	18.9[a]	15.4[b]	2.0	< .02
Gain: feed	.293[a]	.168[b]	.347[c]	.028	< .01
Liver, g	6.60[a]	4.28[b]	6.55[a]	.55	< .01
Liver, % of BW	4.89[a]	4.01[b]	4.66[a]	.33	< .01
Kidney, g	1.12[a]	.97[b]	1.41[c]	.10	< .01
Kidney, % of BW	.83[a]	.91[b]	1.00[c]	.07	< .01
Stomach, g	1.11	1.04	1.14	.13	NS
Stomach, % of BW	.82[a]	.97[b]	.81[a]	.06	< .01
Small intestine, g	5.60[a]	4.51[b]	5.20[a]	.59	< .01
Small intestine, % of BW	4.13[a]	4.24[a]	3.40[b]	.39	< .03
Large intestine, g	1.53[a]	1.59[a]	2.36[b]	.26	< .01
Large intestine, % of BW	1.13[a]	1.49[b]	1.68[c]	.17	< .01

[a,b,c]Means in the same row without a common superscript differ (P < .05).

[d]NS = not significant.

were similar. Liver and kidney absolute and relative weights were increased (P < .01) by HP compared with either C or HF.

Histologic changes in small intestine corresponding to changes observed in relative organ weight were noted in pigs fed HP, and to a lesser extent in pigs fed HF. The changes induced by HF and HP included an increased diameter of the intestinal cross-sectional, with proportional increases in lumen, mucosa and smooth muscle. In addition, there was an underlying inflammatory condition in the ileum of all animals, including the controls, varying in severity between animals and among groups. The inflammation was suggestive of the presence of Campylobacter (Rowland and Lawson, 1985), whose identity was confirmed by the Warthin-Starry strain. The ileal lesions included marked infiltration of lamina propria by mononuclear cells, mainly plasma cells and lymphocytes; many villi appeared shortened with irregular fusion. There was crypt epithelium downgrowth into subjacent lymphoid tissue, lymphoid hyperplasia of Peyer's patches, scattered crypt abscesses and the presence of many goblet cells in the epithelium. The severity of the inflammatory changes was greater in pigs fed HP, suggesting a diet × infection interaction. There was no clinical evidence of Campylobacter infection during the course of the experiment, except that on the day of slaughter

two pigs had severe bloody diarrhea and one pig was found dead in the pen and, on necropsy, was diagnosed as having acute Campylobacter infection. Two pigs fed the HP diet had large polyps in the colon. The surface epithelial component consisted of basophilic columnar cells with variable numbers of mitotic figures; the lamina propria was infiltrated by mononuclear inflammatory cells. Similar polyps have been described in association with Campylobacter infection (Rowland and Lawson, 1985).

Rat Experiment. Daily gain was depressed by HF (P < .01) but was not affected by HP (Table 5). Daily feed intake was reduced by HP, but not by HF, resulting in a lower gain:feed ratio in rats fed HF and a higher ratio in rats fed HP, compared with controls (P < .01).

Absolute and relative (percentage of body weight) weight of the liver was less (P < .01) in rats fed HF than in rats fed C or HP. Absolute weight of kidneys was less in rats fed HF (P < .01) and more in those fed HP (P < . 01) than in controls, but relative weight was greater in rats fed HF (P < .01) than in controls, and that of rats fed HP was greater than of those fed C or HF diets (P < .01). Absolute stomach weight was not affected by diet, but relative weight was greater (P < .01) in rats fed HF than in those fed and HP. Absolute weight of small intestine was less (P

TABLE 6. EFFECT OF DIETARY FIBER AND PROTEIN LEVELS ON GASTROINTESTINAL TRACT DRY WEIGHT AND PERCENTAGE DRY MATTER

	Diet				Probability of
Trait	Control	High fiber	High protein	SD	diet effect
No. of rats	4	4	4		
Dry wt, g					
Stomach	.222	.184	.214	.023	NS[c]
Small intestine	1.255[a]	.865[b]	1.201[a]	.156	< .01
Large intestine	.279[a]	.226[a]	.364[b]	.048	< .01
DM, %					
Stomach	20.51[a]	19.03[b]	19.90[a]	.650	< .03
Small intestine	21.75	20.97	23.69	1.728	NS
Large intestine	18.77[a]	15.85[b]	15.73[b]	1.273	< .01

[a,b]Means in the same row not sharing a common superscript differ ($P < .05$).

[c]NS = not significant.

< .01) in rats fed HF than in C or HP rats, whereas that of large intestine was greater ($P < .01$) in HP than in C or HF rats. Relative small intestine weight was similar in C and HF rats, both of which had a larger mean weight than that of rats fed HP ($P < .03$). Relative weight of the large intestine was increased by HF ($P < .01$) and still further by HP ($P < .01$) compared with controls.

Small intestine dry weight was reduced ($P < .01$) by HF; large intestine dry weight was not affected by HF but was increased by HP ($P < .01$) compared with controls, and percentage of dry matter in the large intestine was less in HF and HP rats ($P < .01$) than in controls (Table 6).

Coliform count was less in HP rats ($P < .05$) than in rats fed HF or the C diet, whereas E. coli count was less ($P < .05$) in HF and HP rats than in C (Table 7). Enterobacteriaceae, Campylobacter and total anaerobe contents were unaffected by diet. Salmonella species were present in rats fed all three diets, whereas Shigella was found only in rats fed HF. The number of cellulolytic microorganisms was least ($P < .05$) in rats fed HF and most ($P < .05$) in those fed HP.

Discussion

Weight gain of swine fed HF was reduced, as expected. This was associated with reduced feed intake and backfat and an increase in trimmed lean cuts as a percentage of body weight. The increased relative weights of kidneys, liver, small intestine and large intestine observed in the present work supports previous observations (Coey and Robinson, 1954; Bohman et al., 1955; Kass et al., 1980; Pond et al., 1988) and, based on the established correlation between empty visceral mass and fasting heat production (Holliday et al., 1967; Holliday, 1971; Baldwin et al., 1980; Koong et al., 1985), provides indirect evidence that HF not only reduces digestible energy intake, but also increases basal metabolic rate of the animal (Pond et al., 1988). Rats fed HF responded similarly to swine in weight gain, gain:feed ratio and relative kidney and large intestine weights. However, relative liver weight was less, rather than more, in rats fed HF fiber than in controls, and small intestine relative weight was unchanged in rats but increased in pigs. The species difference in response of the small intestine and liver to high dietary fiber may be related to differences in relative feed intake (1.3 to 1.6% of body weight daily for swine vs 15 to 20% of body weight daily for rats), to the practice to coprophagy in rats, to other differences in digestive physiology, including the different ratio of glandular to nonglandular stomach in the rat than in the pig, or to differences in stage of growth of rats compared with swine in the present experiment. Rats were in early postweaning growth, whereas swine were near normal slaughter weight, and therefore entering the plateau phase of the growth curve. The relatively low feed intake of pigs in all diet groups was not predicted, and the reason is not apparent. A possible contributing factor is the presence of a subclinical Campylobacter infection as indicated by the histopathological data.

Pigs fed the HP diet had depressed weight gain and feed consumption and increased liver

TABLE 7. MICROBIOLOGICAL PROFILE OF LARGE INTESTINAL CONTENTS OF RATS
FED DIFFERENT DIETS (LOG CFU/GRAM)

Ingredient	Diet			
	Control	High fiber	High protein	SD
Coliforms	6.72[a]	6.56[a]	5.97[b]	.43
Enterobacteriaceae	6.76	6.51	6.45	.36
E. coli	6.75[a]	4.21[b]	3.30[b]	.79
Campylobacter	2.63[a]	1.39[a]	1.42[a]	1.07
Salmonella	+	+	+	
Shigella	−	+	−	
Anaerobes	10.42	10.08	10.56	.17
Cellulolytic microorganisms	7.99[a]	7.43[b]	8.15[c]	.15

[a,b,c]Means within rows with different superscripts differ (P < .05).

and kidney weights compared with pigs fed the C diet. This is in agreement with previous work in growing pigs (Sugahara et al., 1969). Swine and rats responded similarly to HP with respect to relative weight of the large intestine and stomach in that large intestine was increased in both species and stomach weight was unchanged compared with controls. Relative small intestine weight was increased in pigs but decreased in rats fed HP, indicating a species difference in response, perhaps related to coprophagy in rats or to other fundamental differences in digestive physiology. Both dry matter percentage and dry tissue weights were affected by diet in rats. This indicates that turgidity of the tissue was affected by diet, and that tissue accretion of organic matter was increased, at least in rats fed HP. Dry matter was not determined in pig large intestine, but it is assumed that the increased mass in pigs fed HP was associated with increased dry matter accretion.

The physiological basis for the apparent shift in relative sizes of visceral tissues in response to HF or HP diets is unclear. Lipkin (1981) and Johnson (1981) reviewed the knowledge of gastrointestinal cell proliferation and differentiation and regulation of gastrointestinal tract growth, but the specific roles of food intake and composition as controlling factors are unknown. Johnson (1981) described two types of stimulation that result in gastrointestinal mucosal growth:nongastric hormones such as thyroxin and somatotropin and factors associated with food ingestion such as gastrointestinal motility, endocrine and paracrine secretions and neural stimuli. More research is needed to identify and evaluate these factors to allow a more complete understanding of gastrointestinal response to diet.

The identification of Campylobacter in the intestinal tract of pigs raised the important question of whether the apparent hypertrophy of the large intestine was due to inflammation and cellular proliferation in response to the pathogen or the high dietary protein intake of pigs whose large intestinal weight was increased most dramatically. Campylobacter metabolizes amino acids as a primary substrate for growth. The data from the rat experiment provide evidence that the hypertrophic response of the large intestine was due to high dietary protein rather than to Campylobacter, in view of the absence of a change in Campylobacter count in the intestinal contents of rats fed HP, even though large intestine weight was 154% of that of rats fed the C diet. Campylobacter-induced changes were present in the intestine of pigs in all groups. The apparently greater inflammatory response in pigs fed HP than in controls suggests the possibility of a diet × microbe interaction with respect to Campylobacter infection. There is no evidence that performance was adversely affected by the presence of Campylobacter, but the experiment may have ended in the early stages of infection.

No increase in concentration of cellulose-degrading organisms was observed in rats when they were fed the HF diet compared with controls. The probable shorter transit time and larger volume of digesta through the gastrointestinal tract of animals fed HF diets must be considered in interpreting microbial responses to diet when activity is expressed as counts per gram of sample. The results suggest that the rat and pig cellulose-degrading organisms may respond differently to HF diets (Varel, 1987), even though Bacteroides succinogenes and Ruminococcus flavefaciens are presumed to be

the predominant fiber-degrading microorganisms in both the pig and rat (Montgomery and Macy, 1982).

It is concluded that both dietary fiber and protein level have specific effects on visceral organ mass, which may have implications for efficiency of animal growth and energy utilization. In view of the large (30 to 50%) increases in mass of the visceral organs by pigs fed the HP diet, it was surprising that gain:feed ratio was not reduced markedly with this diet. However, changes in gain:feed ratio may not directly reflect changes in energetic efficiency when feed intake or body fat deposition differ.

Literature Cited

Andrews, W. H., P. L. Poelma and C. R. Wilson. 1984. Isolation and identification of Salmonella species. In: Bacteriological Analytical Manual (6th Ed.). Association of Official Analytical Chemists, Washington, DC.

Baldwin, R. L., N. E. Smith, J. Taylor and M. Sharp. 1980. Manipulating metabolic parameters to improve growth rate and milk secretion. J. Anim. Sci. 51:1416.

Bohman, V. R., J. E. Hunter and J. McCormick. 1955. The effect of graded levels of alfalfa and aureomycin upon growing-fattening swine. J. Anim. Sci. 14:499.

Busta, F. F., E. H. Peterson, D. M. Adams, and M. G. Johnson. 1984. Colony count methods. In: M. L. Speck (Ed.) Compendium of Methods for the Microbiological Examination of Foods (2nd Ed.). American Public Health Association, Washington, DC.

Coey, W. E. and K. L. Robinson. 1954. Some effects of dietary crude fiber on live weight gains and carcass conformation of pigs. J. Agric. Sci. (Camb.) 45:41.

Ferrell, C. L. and L. J. Koong. 1986. Influence of plane of nutrition on body composition, organ size and energy utilization of Sprague-Dawley rats. J. Nutr. 116:2525.

Holliday, M. A. 1971. Metabolic rate and organ size during growth from infancy to maturity and during late gestation and early infancy. Pediatrics. 47:169.

Holliday, M. A., P. Potter, J. Jarrah and S. Bearg. 1967. The relation of metabolic rate to body weight and organ size. Pediatr. Res. 1:185.

Johnson, L. R. 1981.Regulation of gastrointestinal growth. In: L. R. Johnson (Ed.) Physiology of the Gastrointestinal Tract. Vol. I. pp 169_196.Raven Press, New York.

Kass, M. L. P. J. Van Soest, W. G. Pond, B. Lewis and R. E. McDowell. 1980. Utilization of dietary fiber from alfalfa by growing swine. I. Apparent digestibility of diet components in specific segments of the gastrointestinal tract. J. Anim. Sci. 50:175.

Koong, L. J., C. L. Ferrell and J. A. Nienaber. 1982a. Effect of plane of nutrition on organ size and fasting heat production in swine and sheep. In: A. Ekern and F. Sundstal (Ed.) Energy Metabolism of Farm Animals. pp 245_248. Proc. 9th Symp., EAAP Publ. No. 29.

Koong, L. J., C. L. Ferrell and J. A. Nienaber. 1985. Assessment of interrelationships among levels of intake and production, organ size and fasting heat production in growing animals. J. Nutr. 115:1383.

Koong, L. J., J. A. Nienaber and H. J. Mersmann. 1983. Effects of plane of nutrition on organ size and fasting heat and production in genetically obese and lean pigs. J. Nutr. 113:1626.

Koong, L. J., J. A. Nienaber, J. C. Pekas and J. T. Yen. 1982b. Effects of plane of nutrition on organ size and fasting heat production in pigs. J. Nutr. 112:1638.

Lipkin, M. 1981. Proliferation and differentiation of gastrointestinal cells in normal and disease states. In: L. R. Johnson (Ed.) Physiology of the Gastrointestinal Tract. Vol. I. pp 145_168.Raven Press, New York.

Mehlman, I. J., W. H. Andrews and B. A. Wentz. 1984. Coliform bacteria. In: Bacteriological Analytical Manual (6th Ed.). Association of Official Analytical Chemists, Washington, DC.

Montgomery, and J. M. Macy. 1982. Characterization of rat cecum cellulolytic bacteria. Appl. Environ. Microbiol. 44:1435.

Pekas, J. C., J. T. Yen and W. G. Pond. 1983. Gastrointestinal, carcass and performance traits of obese versus lean genotype swine: Effects of dietary fiber. Nutr. Rep. Int. 27:259.

Pond, W. G., J. T. Yen and H. J. Mersmann. 1986. Response of the gastrointestinal tract of rats to severe restriction of diet or of nonprotein calories. Nutr. Rep. Int. 33:831.

Pond, W. G., H. G. Jung and V. H. Varel. 1988. Effect of dietary fiber on young genetically lean, obese and contemporary pigs: Body weight, carcass measurements, organ weights and digesta content. J. Anim. Sci. 66:699.

Rowland, A. C. and G.H.K. Lawson. 1985. Intestinal adenomatosis complex (porcine proliferative enteropathies). In: A. D. Leman, B. Straw, R. D. Glock, W. L. Mangeling, R.H.C. Penny and E. Scholl (Ed.) Diseases of Swine (6th Ed.). Chap. 47. pp 547–556. Iowa State Univ. Press, Ames.

SAS. 1985. SAS User's Guide: Statistics. SAS Inst., Inc., Cary, NC.

Sugahara, M., D. H. Baker, B. G. Harmon and A. H. Jensen. 1969. Effect of excess level of dietary crude protein on carcass development in swine. J. Anim. Sci. 40:121.

Varel, V. H. 1987. Activity of fiber-degrading microorganisms in the pig large intestine. J. Anim. Sci. 65:488.

Varel, V. H. and W. G. Pond. 1985. Enumeration and activity of cellulolytic bacteria from gestating swine fed various levels of dietary fiber. Appl. Environ. Microbiol. 49:858.